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MOGAD MAY NOT ONLY MIMIC IMMUNE OR INFECTIOUS ENCEPHALITIS, BUT CAN ALSO BE TRIGGERED BY VIRAL INFECTIONS

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Encephalitis, MOGAD, ADEM, immunoglobulins, SARS-CoV-2

LETTER TO THE EDITOR

We were interested to read the article by Karavassilis et al. on a 16-year-old boy with myelin oligodendrocyte glycoprotein-associated disease (MOGAD), who was initially suspected to have meningoencephalitis but only fully recovered after administration of glucocorticoids, immunoglobulins and double plasma exchange[1]. Initial imaging studies were inconclusive, but a repeat cerebral magnetic resonance imaging (MRI) showed multifocal, enlarging T2 hyperintense lesions supra- and infra-tentorial, suggestive of acute disseminated encephalomyelitis (ADEM)[1]. There was also a hyperintense signal in both optic nerves and an extensive lesion in the myelon, suggestive of myelitis[1]. The study is convincing, but some points need to be discussed.

The first point is that it is unclear whether or not the patient had the initial cerebral computed tomography and MRI with or without contrast. To assess whether he had an enhancing lesion suggestive of infectious or immunologic encephalitis, it would have been mandatory to perform cerebral imaging with contrast at the time of admission. In this context, magnetic resonance venography on admission to exclude venous sinus thrombosis is also missing.

The second point is that it is unclear which viruses the virus panel contained in the cerebrospinal fluid (CSF). Was the patient also tested for SARS-CoV-2 and HIV in the CSF? A negative RT-PCR for SARS-CoV-2 in the throat does not rule out a SARS-CoV-2 infection in other organs, including the brain. As several cases with MOGAD triggered by SARS-CoV-2 have been reported[2], it would have been imperative to confirm or rule out the presence of SARS-CoV-2 in the CSF. It would also have been advisable to rule out HIV infection, as MOGAD has also been reported in association with HIV[3]. An infection with human endogenous retroviruses (HERVs) must also be ruled out, as viral infections in general and HERVs in particular are considered triggers of MOGAD[4].

The third point is that the CSF was not tested for antibodies associated with autoimmune encephalitis (AIE). Since the CSF tests in the index patient were negative for the presence of a virus, it would have been reasonable to consider and rule out AIE. Since specific antibodies can be detected in the CSF in about half of AIE cases, it would have been useful to confirm or exclude this particular differential diagnosis. MOGAD, in particular anti-MOG-associated encephalitis with seizures (FLAMES), has been described in association

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with N-methyl-D-aspartate (NMDAR) antibodies[5]. One limitation in this regard is that no electroencephalogram was recorded in the index patient.

The fourth point is that it remained unclear why the patient was initially given dexamethasone[1]. Were glucocorticoids administered because a Herxheimer–Jarisch reaction was feared, or was there evidence of cerebral oedema?

The fifth point is that the title indicates that the patient had sepsis, but it is unclear by what criteria the sepsis was diagnosed. Was there thrombocytopenia or was the procalcitonin elevated? Did the patient need catecholamines since admission? Was sepsis diagnosed only on the basis of the four criteria of body temperature <36°C or >38°C, heart rate >90 beats/min, respiratory rate >20 beats/min or pCO_2 <4.3 kPa and neutrophilia >12/mm^3 or neutropenia <4/mm^3? Since the opening pressure at the second lumbar puncture was 127 mmHg, we should know whether or not visual impairment was due to papilloedema on funduscopy.

To summarise, this interesting study has limitations that put the results and their interpretation into perspective. Clarification of these weaknesses would strengthen the conclusions and could improve the study. Since MOGAD not only mimics viral encephalitis but can even be triggered by viral infections, it is advisable to comprehensively test MOGAD patients presenting clinically as encephalitis for viruses in the CSF.

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